U.S. Department of Labor

Office of Administrative Law Judges
800 K Street, NW, Suite 400-N
Washington, DC 20001-8002

(202) 693-7300
(202) 693-7365 (FAX)

Issue Date: 21 December 2005

In the Matter of:

RACHEL M. ANDERSON, widow of ROGER ANDERSON
Claimant

v.

KELLYS CREEK RESOURCES/
OLD REPUBLIC INSURANCE GROUP
Employer/Insurer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party in Interest

Case No.: 2002 BLA 401

Appearances: Mr. Graham Swafford, Attorney
Mr. Joseph E. Wolfe, Attorney, On Appeal and Remand
For the Claimant

Ms. Debra L. Fulton, Attorney
Mr. W. William Prochot, Attorney, On Appeal
For the Employer

Before: Richard T. Stansell-Gamm
Administrative Law Judge

**DECISION AND ORDER ON REMAND –
DENIAL OF BENEFITS**

This matter involves a claim filed by Mrs. Rachel M. Anderson for survivor benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("the Act"). Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as "black lung disease."

This Decision and Order on Remand represents my second evaluation of Mrs. Anderson's claim for survivor benefits under the Act. My decision in this case is based on all documents admitted into evidence during the first adjudication of the claim (DX 1 to DX 35, CX

1, and EX 1 and EX2)[1] and the additional medical reports submitted by the parties in conjunction with this remand: CX 2 – Dr. Joshua A. Perper's medical report, dated May 12, 2005; EX 3 – Dr. Richard L. Naeye's medical report, dated August 18, 2005; EX 4 – Dr. Lawrence Repsher's medical report, dated August 24, 2005; and, EX 5 – Dr. Gregory J. Fino's medical report, dated August 31, 2005.

Procedural Background

The procedural background of this case up to the time of the decision by the Benefits Review Board ("BRB" or "Board") was covered in my February 24, 2004 Decision and Order and by the BRB in its January 27, 2005 Decision and Order. Following the BRB's remand of the case, and upon a March 28, 2005 request from Employer's counsel, I issued a Remand Notice, dated April 27, 2005, providing the parties 30 days to submit a brief or statement addressing the issues required to be resolved during the remand. On May 24, 2005, I received additional medical evidence from Claimant's counsel. As a result, I deferred any further action pending a response from Employer's counsel. Subsequently, upon review of the case file and in the absence of any response from the Employer's counsel, I discovered that the copy of the first remand notice to Mr. Prochot had been returned to the Office of Administrative Law Judges due to an incomplete address. To ensure all parties had been placed on adequate notice and had an opportunity to develop additional evidence I issued a second Notice of Remand, dated July 28, 2005, and a Time Extension, dated August 30, 2005, which kept the record open through October 15, 2005. During that period, I received three additional medical reports from Employer's counsel.

At this point, the most important aspects of the procedural history are the previous findings which have been affirmed by the BRB and thus become the "law of the case" and the issue the Board identified as needing to be resolved.

The Board affirmed my determination, based on the parties' stipulation of fact in the hearing before Administrative Law Judge Stuart Levin in September 2001, that Mr. Anderson had 15.13 years of coal mine employment. Next, the BRB affirmed my findings that Mrs. Anderson is an eligible survivor under the Act. Finally, the Board upheld my finding under 20 C.F.R. § 718.202 (a) (2), that the autopsy evidence established the presence of pneumoconiosis arising out of Mr. Anderson's coal mine employment.

On the other hand, the BRB did not affirm my additional finding that the medical opinions of Dr. Morse and Dr. Peacock a) were more probative, and b) established that coal workers' pneumoconiosis hastened Mr. Anderson's death.[2] Consequently, the preponderance of the medical opinion no longer demonstrated that Mr. Anderson's death was due to coal workers' pneumoconiosis.

[1]The parties agreed to have the first decision rendered on the record without a hearing.

[2]Upon remand, the Board also directed for diverse reasons that I re-evaluate my probative weight determinations regarding the opinions by Dr. Naeye and Dr. Fino.

ISSUE ON REMAND

Whether coal workers' pneumoconiosis was a substantially contributing cause of Mr. Anderson's death.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Survivor Claim

To receive survivor benefits under the Act, and the implementing regulations, 20 C.F.R. § 718.205 (a),[3] a surviving spouse must prove by a preponderance of the evidence several facts. First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible for benefits under the Act if she was married to, and living with, the coal miner at the time of his death, and has not remarried.[4]

Next, the claimant must prove the coal miner had pneumoconiosis.[5] "Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment. The regulatory definitions include both clinical pneumoconiosis (the diseases recognized by the medical community as pneumoconiosis) and legal pneumoconiosis (defined by regulation as any chronic lung disease arising out of coal mine employment).[6] The regulation further indicates that a lung disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."[7] As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

Once a determination has been made that a miner had pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.[8] If a miner who was suffering from pneumoconiosis was employed for ten years or more in one or more coal mines, there is a rebuttable presumption that pneumoconiosis arose out

---

[3]Since Mrs. Anderson filed her claim, the U.S. Department of Labor has published new regulations concerning black lung disability benefits. Most of the provisions in Part 718 of those new regulations are applicable to her case.

[4]20 C.F.R. § 718.4 indicates that the definitions in 20 C.F.R. § 725.101 are applicable. 20 C.F.R. § 725.101, in turn, refers to the term "survivor" as used in Subpart B of Part 725. 20 C.F.R. § 725.214 then sets out the spousal relationship requirements and 20 C.F.R. § 725.215 describes the dependency rules. According to § 725.214 (a) the spousal relationship exists if the relationship is a valid marriage under state law. Under § 725.215(a), a spouse is deemed dependent if she was residing with the miner at the time of his death.

[5]20 C.F.R. § 718.205 (a) (1) and see *Trumbo v. Reading Anthracite Co*., 17 B.L.R. 1-85 (1993).

[6]20 C.F.R. § 718.201 (a) (1) and (2).

[7]20 C.F.R. § 718.201 (b).

[8]20 C.F.R. §§ 718.203 (a) and 205 (a) (2).

of such employment.[9] Otherwise, the claimant must provide competent evidence to establish the relationship between pneumoconiosis and coal mine employment.[10]

Finally, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis.[11]

In summary, a survivor claim filed after January 1, 1982 must meet four primary elements for entitlement. The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the survivor claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986) and *Roberts v. Bethlehem Mines Corp*., 8 B.L.R. 1-211 (1985). The four elements are: (1) the claimant is an eligible survivor of the deceased miner; (2) the coal miner suffered from pneumoconiosis; (3) the coal miner's pneumoconiosis arose out of coal mine employment; and, (4) the coal miner's death was due to coal workers' pneumoconiosis.

As noted above, through the BRB's affirmation of my findings, Mrs. Anderson has established the first three requisite elements. Mrs. Anderson is an eligible survivor under the Act and her husband, Mr. Roger Anderson, had coal workers' pneumoconiosis. Consequently, to receive survivor benefits under the Act, Mrs. Anderson must prove by a preponderance of the evidence that Mr. Anderson's death was due to coal workers' pneumoconiosis.

Death Due to Pneumoconiosis

For a survivor claim filed on or after January 1, 1982, the Department of Labor regulations provide four means by which to establish that a coal miner's death was due to coal workers' pneumoconiosis:[12]

1. The miner had complicated pneumoconiosis;

2. Death was caused by pneumoconiosis;

3. Death was caused by complications of pneumoconiosis; or,

4. Pneumoconiosis was a substantially contributing cause or factor leading to the miner's death.

However, a survivor may not receive benefits if the coal miner's death was caused by traumatic injury, or the principal cause of death was a medical condition not related to

[9]20 C.F.R. § 718.203 (b).

[10]20 C.F.R. § 718.203 (c).

[11]20 C.F.R. § 718.205 (a) (3).

[12]20 C.F.R. §§ 718.205 (c) (1), (2), and (3), and 304.

pneumoconiosis, unless evidence establishes that pneumoconiosis was a substantially contributing cause of death.

In its review of my initial decision, the Benefits Review Board affirmed my finding that the record was insufficient to establish the presence of complicated pneumoconiosis. Thus, Mrs. Anderson is not able to prove her husband's death was due to coal workers' pneumoconiosis under 20 C.F.R. § 718.205 (c) (3), through the irrebuttable presumption under 20 C.F.R. § 718.304 associated with the presence of complicated pneumoconiosis. Likewise, the BRB affirmed my conclusion that the medical evidence was insufficient to prove that coal workers' pneumoconiosis directly caused Mr. Anderson's death under 20 C.F.R. § 718.205 (c) (1). Finally, the BRB also noted that I had found the evidence insufficient to demonstrate that complications from coal workers' pneumoconiosis caused his death. Although the Board did not directly affirm that finding, neither party challenged that determination before the BRB and the additional medical provided during this remand proceeding does not alter my conclusion that the medical evidence is insufficient to establish Mr. Anderson passed away due to complications associated with the presence of coal workers' pneumoconiosis in his lungs.

Issue - Pneumoconiosis Was a Substantially Contributing Cause Of, Or Hastened, Death

Even though neither pneumoconiosis nor its complications caused Mr. Anderson's death and he did not have complicated pneumoconiosis, Mrs. Anderson may still be entitled to survivor benefits if pneumoconiosis was a substantially contributing cause of her husband's death. According to 20 C.F.R. § 718.205 (c) (5), "pneumoconiosis is 'a substantially contributing cause' of a miner's death if it hastens the miner's death." Some courts have interpreted that standard to mean the hastening of a miner's death in any way is sufficient.[13] The U.S. Court of Appeals for the Sixth Circuit has recently refined that interpretation by indicating that coal workers' pneumoconiosis "only 'hastens' a death if it does so through a specifically defined process that reduces the miner's life by an estimable time." *Eastover Mining Co. v. Williams,* 338 F.3d 501, 581 (6th Cir. 2003).[14]

With this standard and the admonitions of the BRB in mind, I return to the relevant medical evidence in the record to determine whether coal workers' pneumoconiosis hastened Mr. Anderson's death.[15]

---

[13]*See Shuff v. Cedar Coal Co.*, 967 F.2d 977, 980 (4th Cir. 1992) and *Richardson v. Director, OWCP*, 94 F.3d 164 (4th Cir. 1996).

[14]Previously, as I cited in my initial decision and order, the U.S. Court of Appeals for the Sixth Circuit had concluded that any acceleration of the miner's death attributable to pneumoconiosis met the hasten standard. *Griffith v. Director, OWCP,* 49 F.3d 184, 186 (6th Cir. 1995). Further, I note with interest, that the *Williams* court was dealing with a case of "legal" pneumoconiosis hastening death and a seemingly speculative causation opinion by a treating physician.

[15]With the exception of newly submitted reports by Dr. Perper, Dr. Naeye, Dr. Fino, and Dr. Repsher, these summaries are taken from the February 2004 Decision and Order. Summaries of Mr. Anderson's radiographic history, pulmonary studies, and medical treatment record are also contained in the original decision.

Dr. Charles W. Harlan
(DX 5, DX 24, and DX 25)

Dr. Harlan, board certified in pathology and clinical pathology, performed an autopsy of Mr. Anderson on January 27, 1999. Upon gross examination of the lungs, Dr. Harlan observed "marked anthracosis of the pleural surfaces, with multiple black anthracotic nodules on pleural surfaces, many of which are confluent." A section cut of the lung tissue revealed moderate deposition of anthracotic material throughout the parenchyma; the "areas of deposition form small nodules."

Upon microscopic examination of Mr. Anderson's lung tissue, Dr. Harlan noted vascular dilatation and congestion. He also observed moderate fibrosis thickening of the pleura; associated moderate deposits of black anthracotic material in the pulmonary lymphatics; and, black anthracotic material in the walls of respiratory bronchioles.

Based on his examination, Dr. Harlan diagnosed mild pulmonary emphysema, moderate pulmonary anthracosis (simple pneumoconiosis) and pulmonary congestion.

Again, in January 1999, based on his gross autopsy examination and microscopic biopsy evaluation, Dr. Harlan diagnosed mild pulmonary emphysema, moderate pulmonary anthracosis (simple pneumoconiosis) and pulmonary congestion. He opined that Mr. Anderson "died with chronic obstructive pulmonary disease, consisting of mild pulmonary emphysema and moderate pulmonary anthracosis (simple pneumoconiosis)."

Dr. Richard Naeye
(DX 15, DX 33, and EX 3)

In October 1999, Dr. Richard Naeye, a board-certified pathologist, reviewed the autopsy slides. Under the microscope, Dr. Naeye saw "a very small amount of black pigment admixed with fibrous tissue." He also reported mild to moderate centrilobular emphysema. Based on his microscopic study, Dr. Naeye diagnosed mild, simple coal workers pneumoconiosis based on the presence of multiple anthracotic macules. Additionally, Dr. Naeye observed that the "walls of small and larger pulmonary arteries have walls nearly as thick as the wall of such vessels in the systemic circulation."

In addition to reviewing the autopsy slides in October 1999, Dr. Naeye also reviewed all medical records that had been admitted into the record to the date of his evaluation. Initially, Dr. Naeye noted that Mr. Anderson had about 17 years of coal mine employment. He had smoked about a pack and a half of cigarettes a day for 30 years and continued to smoke after developing severe pulmonary problems. While showing a dilated right ventricle, the chest x-rays were negative for pneumoconiosis. A pulmonary function test in a 1991 test revealed a mild to moderately severe airway obstruction. Other pulmonary studies produced varying results showing improvement following steroid and antibiotic therapy. The results of the arterial blood gas studies fluctuated between 1991 and 1999.

Although Mr. Anderson had simple coal workers' pneumoconiosis, for several reasons, Dr. Naeye concluded black lung disease played no role in his death. First, Mr. Anderson had a "mild" case of pneumoconiosis. While the lung tissue slides contained "several" micro nodules, Dr. Naeye believed the biopsy samples were not indicative of the profusion of these lesions throughout Mr. Anderson's lungs because the chest x-rays were consistently negative.

Second, and closely related, the focal emphysema associated with the coal workers' pneumoconiosis represented less than 1% of the total emphysema in the tissue samples. If the coal workers' pneumoconiosis lesions had been in sufficient number to adversely affect the pulmonary test results, the lesions would have appeared on the chest x-rays. Consequently, Mr. Anderson's coal workers' emphysema would not have impacted his lung functions.

Third, the remaining centrilobular emphysema, which according to numerous medical studies is unrelated to the inhalation of coal dust, likewise was not severe enough to explain the abnormal pulmonary function tests. Instead, the changing severity of Mr. Anderson's chronic bronchitis and bronchiolitis explain the varying test results. Both pulmonary problems were caused by his cigarette smoking.

Fourth, Mr. Anderson also had a history of pulmonary arterial emboli which likely contributed to his fluctuating hypoxemia. The 1995 pulmonary arterial emboli "apparently produced a permanent increase in pulmonary vascular resistance with resulting cor pulmonale" as evidenced by "the very thick muscular walls of pulmonary arteries."

Fifth, both the centrilobular emphysema and pulmonary arterial emboli permanently increased pulmonary resistance to the extent that Mr. Anderson developed cor pulmonale, as demonstrated by the thick pulmonary artery walls.

In summary, Dr. Naeye concluded Mr. Anderson's coal workers' pneumoconiosis was too mild to have caused any measurable impairment of his lungs, shortened his life, or contributed to his death in any manner. His death can be attributed in large part to damage to the microcirculation of his heart caused by cigarette smoking and by the pulmonary arterial emboli that were the cause of his chronic cor pulmonale. Neither condition has any origin in occupational exposures to coal mine dust.

In June of 2002, Dr. Naeye supplemented his 1999 report after reviewing hospitalization reports from the last days of Mr. Anderson's life. Noting that in the last three months of his life, Mr. Anderson "experienced a rapid downhill course in terms of lung function," Dr. Naeye had "no doubt" that the miner had "the findings of mild, simple coal worker's pneumoconiosis in his lungs at postmortem examination." Mr. Anderson's lungs also contained mild to moderate centrilobular emphysema. However, neither pulmonary problem "was nearly severe enough to have any measurable role in the events that led to the progressive deterioration of his lung function and death." Instead, Dr. Naeye focused on the presence of pulmonary arterial emboli between 1991 and 1995, which caused "severe, permanent pulmonary arterial hypertension as evidenced by the very thick walls of arteries of all sizes in his lungs." Dr. Naeye observed the thick arterial walls in the autopsy slides and reported the result in his 1999 report. According to Dr. Naeye, this condition frequently leads to progressive heart failure and early death. Another

feature of this condition, which is consistent with Mr. Anderson's medical record, is wide fluctuations in blood gas values and varying episodes of dyspnea and associated pulmonary infections such as bronchitis that do not respond to antibiotics or bronchodilators. Consequently, Dr. Naeye concluded Mr. Anderson had "idiopathic pulmonary arterial hypertension" or "repeat pulmonary arterial emboli." This pulmonary disorder does not have an occupational origin. In closing, Dr. Naeye repeated his opinion that Mr. Anderson's pneumoconiosis was "too mild to have had any role in his progressive disability and death."

In an August 18, 2005 supplemental report, Dr. Naeye offered responsive critiques to Dr. Perper's comments about his earlier reports. In assessing the overall severity of Mr. Anderson's coal workers' pneumoconiosis, Dr. Perper failed to cross-check his pathology findings with the radiographic record. Significantly, almost all of the chest x-ray interpretations were negative for the presence of pneumoconiosis. Mr. Anderson's radiographic record effectively excludes the presence of coal workers' pneumoconiosis severe enough to have caused disability or contributed to his death. The pneumoconiosis lesions identified in the pathology study were "very small" and located in areas that "have little or no influence on lung function." The largest lesion measured 1.2 mm, rather than the 2.0 mm reported by Dr. Perper. Dr. Naeye emphasized that besides lung cancer and sudden cardiac arrhythmia, centrilobular emphysema is a major cause of premature death in cigarette smokers. Additionally, the interstitial fibrosis identified by Dr. Perper is really the fused segments of alveolar walls due to emphysema. Finally, the thickening artery walls were not associated with coal dust exposure. Instead, they were a manifestation of idiopathic pulmonary artery hypertension which played a major role in Mr. Anderson's death.

Dr. John C. Morse
(DX 4, DX 14, DX 24, DX 25, DX 31, and CX 1)

Dr. Morse, board certified in family practice, examined and treated Mr. Anderson on numerous occasions for multiple ailments at the Horizon Medical Center in Dickson, Tennessee from about January 1996 to the time of his death three years later. Many of Mr. Anderson's presenting complaints involved pulmonary distress which led Dr. Morse to refer Mr. Anderson to Dr. Peacock. Throughout this period, Dr. Morse heard persistent wheezing and decreased breath sounds. Dr. Morse prescribed several medications for the patient's respiratory distress, including supplemental oxygen and inhalers. On a couple of occasions, Mr. Anderson was hospitalized for pneumonia. Dr. Morse's consistent pulmonary diagnoses were chronic obstructive pulmonary disease and pneumoconiosis. In October of 1998, Dr. Morse reported that the miner's pulmonary condition was slightly improved, but "probably as good as it will ever get," even though he was on oxygen.

Towards the end of January 1999, Mr. Anderson was being treated in the acute care and ICU sections of Horizon Medical Center for shortness of breath. On January 26, 1999, he was transferred to the Subacute unit. On that day, Mr. Anderson had become "somewhat dull" mentally and his oxygen saturation dropped. The next day, January 27, 1999, at 8:30 a.m., Dr. Morse was called by the hospital personnel and informed of Mr. Anderson's falling oxygen saturation. He directed Mr. Anderson's transfer to ICU and proceeded to the hospital. When Mr. Anderson presented at ICU, he displayed "some focal seizure activity." As a result, Dr.

Huffnagle, a neurologist, responded. After Dr. Morse arrived, Mr. Anderson's oxygen saturation improved; however, the improvement was only brief. Mr. Anderson was intubated but then developed "junctional rhythm." Dr. Chambers, a cardiologist, responded. In the presence of Dr. Morse, Dr. Huffnagle, and Dr. Chambers, Mr. Anderson went into cardiac arrest. For about the next hour, the physicians attempted to resuscitate Mr. Anderson with CPR and the use of a temporary pacemaker. Their efforts were unsuccessful and Mr. Anderson expired at 9:30 a.m. In the closing of the final discharge summary, Dr. Morse stated, "It was felt that his cause of death was his underlying black lung pneumoconiosis." The final discharge diagnoses were: coal worker's pneumoconiosis (black lung disease), obstructive chronic bronchitis, congestive heart failure, convulsions, cardiac arrest, diabetes, and bipolar affective disorder.

On February 4, 1999, Dr. Morse signed Mr. Anderson's death certificate documenting that Mr. Anderson died on January 27, 1999, at the age of 49. Dr. Morse believed his death was due to coal miner's black lung disease which he obtained by working in coal mines. Dr. Morse left blank the portion of the death certificate concerning whether an autopsy had been accomplished.

In August of 2000, Dr. Morse completed a questionnaire with generally terse answers indicating that he had treated Mr. Anderson from 1995 until his death for chronic obstructive pulmonary disease. Additionally, an echocardiogram, while showing "good" heart function, established the presence of cor pulmonale. The physician had also treated Mr. Anderson for pneumonia. Through "chest x-ray," Dr. Morse concluded Mr. Anderson had pneumoconiosis. Prior to his death, Mr. Anderson was totally disabled due to pneumoconiosis. In response to the question whether Mr. Anderson died as a result of pneumoconiosis "and/or" whether pneumoconiosis was a substantial contributing cause "and/or" factor leading to his death, Dr. Morse replied, "Yes." He provided no further information.

Dr. Mark D. Peacock
(DX 6, DX 14, CX 19, DX 24, DX 25, and CX 1)

Dr. Mark Peacock, board certified in pulmonary disease and internal medicine, evaluated Mr. Anderson for his pulmonary disorder on April 12, 1996. Mr. Anderson presented with a "past diagnosis of pneumoconiosis secondary to exposure to coal dust and a history of emphysema." Mr. Anderson was on continuous oxygen therapy and struggling with shortness of breath. He reported that his brother had severe emphysema. Mr. Anderson stopped smoking cigarettes a year ago. Mr. Anderson had also been informed that the right side of his heart was enlarged due to his pulmonary compromise. In March 1995, he suffered a pulmonary embolus. Upon examination, the physician heard bilateral dry crackles and expiratory wheezes. A March 26, 1996 chest x-ray showed bilateral interstitial disease with a slightly increased heart silhouette. The physician found oxygen desaturation upon exercise and observed an irregular heart beat. According to the doctor, Mr. Anderson presented a complicated pulmonary situation. He had significant pulmonary emphysema, interstitial lung disease, "presumed secondary to coal workers' pneumoconiosis," history of pulmonary embolus, and "probable pulmonary hypertension with right ventricular hypertrophy." Dr. Peacock concluded Mr. Anderson was a young male with a "very severe pulmonary compromise due to a combination of severe emphysema and a [sic] probable coal workers' pneumoconiosis."

In a May 1996 letter, stating the results of pulmonary function tests, Dr. Peacock diagnosed COPD (chronic obstructive pulmonary disease) and indicated that Mr. Anderson was totally disabled as a coal miner under Department of Labor standards. In the same letter, Dr. Peacock also noted that Mr. Anderson had "a history of pneumoconiosis presumed secondary to his exposure as a coal worker."

Between October 1996 and February 1997, Dr. Peacock evaluated Mr. Anderson on several occasions for problems associated with his COPD. On one occasion, Mr. Anderson experienced exacerbation of his pulmonary disease which required emergency room treatment. Dr. Peacock noted the presence of interstitial lung disease in Mr. Anderson's chest x-rays, which he suspected was secondary to coal workers' pneumoconiosis.

In September 1997, Dr. Peacock evaluated Mr. Anderson for continued shortness of breath. He again found diffuse expiratory wheezes and mild basilar crackles. Dr. Peacock diagnosed COPD and pneumoconiosis.

Two months later, in November 1997, Dr. Peacock conducted sleep disorder tests. Having noted that Mr. Anderson had "advanced emphysema and interstitial lung disease due to pneumoconiosis," Dr. Peacock stated the tests established Mr. Anderson also had "moderate obstructive sleep apnea." Dr. Peacock believed his condition was "complicated by baseline pulmonary lung disease with frequent O_2 desaturations."

In an April 1998 letter, Dr. Peacock indicated that he had treated Mr. Anderson over the course of two and a half years. In his opinion, Mr. Anderson suffered from "progressive pulmonary dysfunction due to both obstructive lung disease and interstitial fibrosis." Mr. Anderson would have a "chronic life-long disability with severe dyspnea on exertion due to his ongoing lung problems" and was "permanently disabled with regards to active employment."

Between April 28, 1998 and November 17, 1998, Dr. Peacock examined Mr. Anderson four times for exacerbation of COPD/bronchitis. Upon examination, he noted both inspiratory crackles and expiratory wheezing. Dr. Peacock diagnosed COPD. Mr. Anderson was unable to breathe without continuous oxygen therapy. He was taking steroids which provided some relief.

In October of 2000, Dr. Peacock responded to a questionnaire about Mr. Anderson's pulmonary condition. According to Dr. Peacock, over the course of two and a half years, he treated the miner for COPD and had prescribed inhalers, steroids and other medicine for his pulmonary problems. In his examinations of Mr. Anderson's lungs, Dr. Peacock consistently heard wheezing and crackling. Mr. Anderson presented to Dr. Peacock with a history of pneumoconiosis; nothing in his evaluations had "discredited" that pre-existing diagnosis. Mr. Anderson was totally disabled at the time of his death. In response to the question whether Mr. Anderson died as a result of pneumoconiosis "and/or" whether pneumoconiosis was a substantial contributing cause "and/or" factor leading to his death, Dr. Peacock replied, "Yes." He provided no further information.

Dr. David E. Chambers
(DX 24, DX 25, and DX 31)

On March 9, 1995, Mr. Anderson arrived for a cardiac consult examination with Dr. Chambers, who was board certified in cardiovascular disease and internal medicine, due to shortness of breath and fatigue. Because he appeared to be in respiratory distress at that time, Mr. Anderson was admitted to the hospital for treatment and evaluation. The admission diagnosis was pulmonary embolus. Nine days later, Mr. Anderson was discharged from the hospital. A CT study had confirmed a pulmonary embolus and Mr. Anderson was successfully treated for the condition.

Between March 1995 and August 1995, Dr. Chambers conducted several follow-up evaluations of Mr. Anderson's condition. Mr. Anderson had experienced multiple pulmonary emboli. However, his pulmonary condition had stabilized. Specifically, Dr. Chambers informed Mr. Anderson on April 3, 1995 that many of the blood clots in his lungs had dissolved and that his lungs were improving. Collaterally, Dr. Chambers diagnosed "end stage emphysema, COPD, and lung damage."

In an April 18, 1995 letter, Dr. Chambers expressed his opinion that Mr. Anderson had black lung disease. Dr. Chambers based his opinion on the following information: medical records, awareness of Mr. Anderson's coal mine employment, pulmonary function tests showing severe obstructive pulmonary disease, and a chest x-ray.

After a hospitalization for pneumonia, Mr. Anderson saw Dr. Chambers in September 1995 for an evaluation. Mr. Anderson reported improvement in his condition. Dr. Chambers heard diffuse rhonchi and suggested Mr. Anderson continue his oxygen therapy.

In an October 1995 follow-up examination, Dr. Chambers noted that Mr. Anderson's cardio-pulmonary condition had improved and stabilized. His chest was clear. Dr. Chambers recommended Mr. Anderson continue his medication, return to Dr. Morse for routine health issues and seek emergency treatment for any shortness of breath, chest pain or fever.

On January 27, 1999, Dr. Chambers was called to ICU as Mr. Anderson went into cardiac arrest. Mr. Anderson had a history of seizure disorder and severe lung disease. Upon arrival, Mr. Anderson was not breathing, his cardiac rate was asystole, and his blood pressure was zero. Mr. Anderson was intubated and full CPR was continued. A venous pacemaker was inserted and a cardiac rhythm was established. However, there was no blood pressure or respiratory effort. A portable chest x-ray showed no significant cardiomegaly or pneumothorax. When Mr. Anderson did not revive, CPR efforts were terminated at 9:32 a.m.

Dr. Vera Huffnagle
(DX 31)

On January 27, 1999, Dr. Huffnagle, a board certified neurologist, was called to ICU to evaluate Mr. Anderson's neurological condition. She noted that Mr. Anderson had a history of black lung. For the past months, he struggled with "intermittent bouts of right-sided head

twitching" which was "possibly secondary" to his medication for a mental condition. Mr. Anderson had been hospitalized since January 21, 1999 with oxygen therapy and steroids for shortness of breath and exacerbation of COPD. A day or two earlier, Mr. Anderson was complaining about a headache and had become lethargic. When Dr. Huffnagle was called to ICU, Mr. Anderson exhibited right hand and head tremor. Although awake and "breathing comfortably," Mr. Anderson did not respond to commands. Dr. Huffnagle diagnosed a possible seizure disorder and unsuccessfully attempted to stop the tremors with medication. "Approximately seven minutes later he went into respiratory arrest and code was called when the patient was in a junctional rhythm." Even with 100% oxygen, Mr. Anderson's saturation "remained poor." After 55 minutes, Mr. Anderson passed away. Dr. Huffnagle diagnosed a "probable" focal seizure "possibly secondary to a cerebral lesion." The neurological symptoms could be due to cerebral hemorrhage, an infarction, or hypoxemia. Without imaging studies or an autopsy, Dr. Huffnagle could not be more certain.

Dr. Gregory J. Fino
(DX 19, DX 26, EX 1, EX 2, and EX 5)

In February 1997, Dr. Fino, board certified in pulmonary disease and internal medicine, reviewed Mr. Anderson's medical record. As early as 1991, Mr. Anderson was diagnosed with a mild to moderate chronic obstructive pulmonary disease and bronchitis within the context of his continued cigarette smoking. Upon completion of his review, Dr. Fino highlighted Mr. Anderson's high concentration of carbon dioxide in his blood, which is "unusual" for coal workers' pneumoconiosis unless there is significant pulmonary fibrosis in the form of complicated pneumoconiosis. Likewise, Mr. Anderson's severe pulmonary obstruction is inconsistent with pneumoconiosis since coal workers' pneumoconiosis does not cause a totally disabling obstructive impairment. Dr. Fino concluded Mr. Anderson had severe chronic obstructive bronchitis associated with his cigarette smoking. The evidence was insufficient to diagnose simple coal workers' pneumoconiosis.

In December 2000, Dr. Fino completed another report after reviewing the medical record and the autopsy evaluations by Dr. Harlan and Dr. Naeye. In light of the autopsy evidence, Dr. Fino changed his opinion about the presence of black lung disease. He now concluded "there was evidence of pathologic pneumoconiosis." Nevertheless, Dr. Fino remained adamant that Mr. Anderson's severe chronic obstruction was related solely to his cigarette smoking; "coal dust inhalation played no role in this man's severe chronic obstruction."

To support his position, Dr. Fino stated a pulmonary obstruction due to coal dust inhalation could be distinguished from a respiratory obstruction caused by cigarette smoking. When coal dust inhalation causes a clinical pulmonary obstruction, the condition is associated with severe coal dust-related pulmonary fibrosis and displays both obstructive and restrictive defects. Dr. Fino acknowledged some medical studies found a link between coal dust inhalation and a clinical decrease in FEV1. However, citing numerous methodology errors, such as selective subjects, Dr. Fino discounted those findings. According to Dr. Fino, no evidence existed to conclude "there is a clinically significant reduction in the FEV1 as a result of chronic obstructive lung disease due to coal mine dust inhalation." After stating it was well known that complicated pneumoconiosis could cause clinical emphysema, Dr. Fino likewise disputed the

proposition that simple coal workers' pneumoconiosis alone could cause "clinically significant" emphysema. He referenced several studies finding insufficient evidence to conclude that, in the absence of pulmonary massive fibrosis, the incidence of disabling emphysema was higher for coal miners than the general population.

Concerning the cause of Mr. Anderson's death, Dr. Fino found the autopsy to be insufficient because the examination was limited to the lungs. Thus, while the autopsy established the presence of emphysema and coal workers' pneumoconiosis, Dr. Fino believed its limited nature made it "impossible to discern" the cause of death. "We have no idea whether he had a heart attack, or bleeding into his abdomen, or a massive stroke." Consequently, due to the limited nature of the autopsy, Dr. Fino stated there was insufficient evidence to conclude that "coal dust inhalation, or for that matter cigarette smoking related lung disease," either caused or contributed to Mr. Anderson's death.

In September of 2002, Dr. Fino reviewed additional medical evidence, including new x-ray readings developed in January 1999 during Mr. Anderson's last hospitalizations. Upon completion of his review, Dr. Fino stated "the additional medical evidence has not caused me to change any of my opinions."

In an August 30, 2005 report, Dr. Fino indicated that he had reviewed the recent reports by Dr. Perper, Dr. Naeye, and Dr. Repsher. While acknowledging the difference of medical opinions, Dr. Fino remained convinced that Mr. Anderson's inhalation of coal dust played no role in his death. Although pathologic pneumoconiosis was present in Mr. Anderson's lungs, his extensive cigarette smoking history was the cause of his severe COPD. Both cigarette smoke and coal dust can cause significant obstructive lung disease. However, Dr. Fino found sufficient evidence in the record to "determine the degree of both emphysema and chronic obstructive bronchitis caused by cigarette smoking versus coal dust inhalation." In Mr. Anderson's case, clinical evidence indicates the cigarette smoking was the cause of his emphysema rather than coal mine dust. Relying on several medical studies from 1984 though 1995,[16] Dr. Fino noted that the presence of focal emphysema due to coal dust does not necessarily translate into clinical symptoms of emphysema. Only in the presence of significantly increasing pneumoconiosis does the coal dust-related emphysema become significant. Consequently, while coal dust can cause emphysema, the more probative medical studies indicated coal dust-related emphysema does not lead to a significant impairment or disability

Dr. Lawrence H. Repsher
(EX 4)

On August 24, 2005, Dr. Repsher, board certified in pulmonary disease, internal medicine and critical care, conducted a review of the medical evidence in Mr. Anderson's case, including Dr. Harlan's autopsy reports and the reports of Dr. Morse, Dr. Peacock, Dr. Naeye, and Dr. Perper.

[16]Dr. Fino also mentioned a couple of studies that reached contrary conclusions but discounted their conclusions due to faulty methodology.

As background, Dr. Repsher observed that Mr. Anderson had smoked a pack and a half of cigarettes between the ages of 15 to 46. When he stopped smoking in 1995, Mr. Anderson had a cigarette smoking history of 60 pack years.[17] Mr. Anderson had also worked in coal mines for 17 years. In January 1999, Mr. Anderson died after suffering respiratory and then cardiac failure due to the administration of a pulmonary depressant.

According to Dr. Repsher, a coal miner without radiographic evidence of pneumoconiosis usually has a normal lung function. Since Mr. Anderson's radiographic studies did not show the presence of pneumoconiosis and the pathology findings indicated only mild coal workers' pneumoconiosis, the presence of coal workers' pneumoconiosis in his lungs wouldn't have had "any clinically significant impairment of his lung function." Instead, Mr. Anderson's lifetime breathing problems were attributable to his moderately severe cigarette smoking induced COPD.

To support his conclusion, Dr. Repsher referenced medical studies that indicate cigarette smoking is the most common and powerful cause of COPD and centrilobular emphysema. Although coal miners may develop COPD, on average, miners who are non-smokers with only simple pneumoconiosis will have normal lung function. In other words, though coal dust inhalation can cause clinical airways obstruction, in Mr. Anderson's case, his COPD was due to cigarette smoking and not coal dust.

In Dr. Repsher's opinion, Mr. Anderson's mild, clinically insignificant histological pneumoconiosis, which was present in only a small percentage of the pulmonary tissue, would not have caused "any measurable impairment of lung function." Correspondingly, Mr. Anderson's death was not due to coal workers' pneumoconiosis or coal dust inhalation.

In regards to the studies and text cited by Dr. Perper, Dr. Repsher found several flaws. First, most of the studies could be disregarded due to "poor design, inadequate methodology and failure to control the cigarette smoking factor." Second, when Dr. Perper took a passage from a pathology text, he failed to mention the comments preceding the quote which indicates that generally simple coal workers' pneumoconiosis is not associated with symptoms of clinical significance or a reduction in longevity. Third, though the cited study shows that coal dust can cause a chronic airways obstruction, the remaining portion of the data further demonstrates that the only significant cause of airways obstruction is cigarette smoking; coal dust exposure was clinically insignificant.

Dr. Joshua A. Perper
(CX 2)

On May 12, 2005, Dr. Perper, board certified in anatomic, surgical and forensic pathology, conducted a review of Mr. Anderson's extensive medical record, reviewed a lung pathology slide and discussed the cause and nature of Mr. Anderson's death. As part of his review, Dr. Perper considered Dr. Harlan's autopsy report, reviewed the radiographic record and evaluated the medical reports from various physicians, including Dr. Fino and Dr. Naeye. Dr.

[17]A pack year equals the consumption of one pack of cigarettes per day for one year.

Perper noted that Mr. Anderson had cigarette smoking history of 50 pack years and worked as a coal miner for 18 years.

After noting that "a single duplicate lung section is not sufficient for evaluating satisfactorily the severity of the coal workers' pneumoconiosis," Dr. Perper microscopically examined the sole lung specimen. He discovered anthracotic deposits with silica crystals. Also present were multiple, mixed coal dust fibro-anthracotic nodules, scattered throughout the lung tissue. The largest nodule was 2 millimeters. Dr. Perper also observed moderate to severe centrilobular emphysema and marked interstitial fibrosis. Based on his pathology examination, Dr. Perper diagnosed slight to moderate simple, coal workers' pneumoconiosis, macular and micronodular and moderate to severe centrilobular emphysema.

Upon review of the radiographic record, Dr. Perper questioned the meaning of the chest x-ray findings. Many of the physicians believed the notable pulmonary nodules were granulomas. However, in light of the pathologic absence of any granulomas, Dr. Perper believed the "granulomas were in fact lesions of coal workers' pneumoconiosis."

Concerning Dr. Naeye's evaluation, Dr. Perper stated Dr. Naeye had failed to recognize Mr. Anderson's severe pulmonary problems which were consistent with a severe obstructive pulmonary disease. Additionally, he disagreed with Dr. Naeye about the etiology of the pulmonary hypertension. Rather than being idiopathic, the pulmonary hypertension was due to emphysema. Similarly, Dr. Perper disagreed with Dr. Fino's assessment about the cause of centrilobular emphysema. According to Dr. Perper, the presence of centrilobular emphysema was due to both coal dust and cigarette smoke, which can't be differentiated. Further, Dr. Fino's suggested causes of death have little support since the physician apparently didn't consider the clinical evidence of worsening oxygen saturation just prior to Mr. Anderson's death and the treating physician's diagnosis of hypoxemia.

Dr. Perper opined that Mr. Anderson had severe coal workers' pneumoconiosis based on several factors. First, due to the absence of identifiable granulomas in the pathology studies, the radiographic films showed the presence of pneumoconiosis lesions. Second, Mr. Anderson suffered a progressive and worsening respiratory disease that did not respond to treatment. Third, his microscopic evaluation and the autopsy established the presence of significant simple coal workers' pneumoconiosis. Fourth, the pulmonary damage due to coal workers' pneumoconiosis may continue after the cessation of occupational exposure. Fifth, while a known complication of cigarette smoking, centrilobular emphysema may also be caused by coal dust. Medical studies have shown that coal miners develop more emphysema than non-miners with similar cigarette smoking histories.

According to Dr. Perper, based on the length of his coal mine employment, clinical documentation of pulmonary problems and hypertension, and the pathology finding, coal workers' pneumoconiosis was a substantial cause of Mr. Anderson's death and a "hastening factor of his demise." Dr. Perper explained that the presence of pneumoconiosis caused pulmonary insufficiency and hypoxemia by "direct and [sic] replacement of normal lung tissue by non-breathing pneumoconiotic lesions and associated centrilobular emphysema with

pulmonary hypertension and cor pulmonale." Such hypoxemia precipitated and aggravated Mr. Anderson cardiac arrhythmia associated with his heart disease.

Discussion

The treating physicians and medical experts disagree on whether the coal workers' pneumoconiosis in Mr. Anderson's lungs hastened his death.[18] Dr. Morse, Dr. Peacock and Dr. Perper opined that the coal workers' pneumoconiosis present in Mr. Anderson's lungs contributed to, and hastened, his death. Dr. Naeye, Dr, Fino, and Dr. Repsher disagree; they concluded coal workers' pneumoconiosis was not a contributing factor in his death. Due to this conflict in medical opinion, I must first determine the relative probative weight of each assessment and then consider whether the preponderance of the more probative evidence establishes the substantially contributing cause issue.

To have probative value, a medical opinion must be both documented and reasoned. As to the first probative value factor, a medical opinion is likely to be more comprehensive and probative if it is based on extensive objective medical documentation such as radiographic tests and physical examinations. *Hoffman v. B & G Construction Co*., 8 B.L.R. 1-65 (1985). In other words, a medical practitioner who considers an array of medical documentation that is both long (involving comprehensive testing) and deep (includes both the most recent medical information and past medical tests) is in a better position to present a more probative assessment than the physician who bases a diagnosis on a test or two and one encounter.

The second factor affecting relative probative value, reasoning, involves an evaluation of the connections a medical practitioner makes based on the documentation before him or her. Reasoning that is both supported by objective medical tests and consistent with all the documentation in the record, is entitled to greater probative weight. *Fields v. Island Creek Coal Co*., 10 B.L.R. 1-19 (1987). Additionally, to be considered well reasoned, the practitioner's conclusion must be stated without equivocation or vagueness. *Justice v. Island Creek Coal Co*., 11 B.L.R. 1-91 (1988).

With these probative factors in mind, I first note the Benefits Review Board's determination concerning my prior finding about the probative value of Dr. Morse's opinion essentially controls my present assessment of its probative value because the physician has not provided any additional statement during this remand. Dr. Morse treated Mr. Anderson over the last few years of his life for his pulmonary and cardiac health problems and was with Mr. Anderson, attempting to revive him, when he passed away due cardiac arrest. In the final discharge note, Dr. Morse indicated that the cause of death was the underlying black lung disease and signed the death certificate stating cause of death was pneumoconiosis. When queried on a form whether pneumoconiosis was a substantial contributing cause of death, Dr. Morse said yes. According to the BRB's interpretation of the *Williams* case, that affirmed response, without any explanation, does <u>not</u> support a finding that coal workers' pneumoconiosis hastened Mr.

[18]Although he concluded that Mr. Anderson died <u>with</u> moderate pulmonary anthracosis, Dr. Harlan, who conducted the autopsy, did not provide any opinion as to the role the disease played in Mr. Anderson's death. Similarly, while Dr. Chambers and Dr. Huffnagle were attending to Mr. Anderson's cardiac and neurological problems when he died, neither doctor offered an opinion on the cause of death.

Anderson's death. Since Dr. Morse still has not provided a statement about the specific mechanics by which pneumoconiosis hastened Mr. Anderson's life, his medical opinion has diminished probative value and remains insufficient to support a finding of death due to pneumoconiosis.

Essentially, the same probative finding applies to Dr. Peacock's conclusion. Dr. Peacock, a pulmonary specialist, treated Mr. Anderson during his last years for a "very severe pulmonary compromise" due to severe emphysema and probable coal workers' pneumoconiosis. According to Dr. Peacock, at the time of his death, Mr. Anderson was totally disabled and unable to breath without continuous oxygen. When asked whether coal workers' pneumoconiosis was a substantial contributing cause of Mr. Anderson's death, Dr. Peacock replied yes, without further explanation. To date, Dr. Peacock has provided no additional explanation for his affirmative response. Accordingly, for the reasons noted above, Dr. Peacock's answer, without any additional comments, has little probative value on the causation issue.

Based on a well documented medical review and his microscopic examination of lung tissue, Dr. Perper, a well qualified forensic pathologist, opined that coal workers' pneumoconiosis contributed to, and hastened, Mr. Anderson's death through a specific process. The extensive coal workers' pneumoconiosis lesions and associated emphysema in Mr. Anderson's lungs replaced normal lung tissue with non-functioning tissue, which caused pulmonary insufficiency. In turn, the pulmonary insufficiency precipitated and aggravated Mr. Anderson's cardiac arrhythmia.

Although Dr. Perper's explanation may satisfy the *Williams* court's specificity requirement, two significant reasoning issues undermine the probative value of his assessment. First, As Dr. Perper readily acknowledged, his examination of one lung tissue slide, with a finding of "slight to moderate" coal workers' pneumoconiosis, was an insufficient basis upon which to base a conclusion about the extent, or severity, of coal workers' pneumoconiosis in Mr. Anderson's lungs. Nevertheless, Dr. Perper diagnosed "severe" pneumoconiosis in part based on his opinion that the chest x-rays showed extensive pneumoconiosis because the granulomas noted by the physicians reviewing the films were really pneumoconiosis lesions. He reasoned that since the pathology examination did not disclose the presence of any granulomas, the noted opacities were incorrectly diagnosed as granulomas, rather than pneumoconiosis.

However, Dr. Perper's reliance on such radiographic evidence to support his causation conclusion is problematic. As I previously noted, the BRB affirmed my prior determination that the preponderance of the chest x-ray evidence is insufficient to establish the presence of pneumoconiosis. Consequently, Dr. Perper's opinion that the radiographic films show extensive coal workers' pneumoconiosis is inconsistent with my affirmed determination to the contrary, which is the law of the case. Additionally, Dr. Perper's reasoning in this regard has little persuasive merit. Again, Dr. Perper only looked at one lung tissue sample and the other pathologists only reviewed tissue samples taken from the darkened pigmented areas of Mr. Anderson's lungs. There is no indication in the record that an attempt was made to specifically identify the granulomas. Further, even if the pathology findings medically impeached the chest x-ray interpretations, those purported misinterpretations do not necessarily mean the nodules in the chest x-rays must be pneumoconiosis lesions. Finally, Dr. Perper only reviewed the chest x-

ray reports; he did not see the actual films. In contrast, none of the highly qualified board certified radiologists who were also B readers and actually viewed Mr. Anderson's chest x-rays saw any evidence of nodules consistent with pneumoconiosis.[19]

Second, Dr. Perper also supported his diagnosis of severe coal workers' pneumoconiosis on the basis that the centrilobular emphysema in Mr. Anderson's lungs may in part be attributable to his exposure to coal dust. To support his assessment on this condition, Dr. Perper cited several medical studies. While I have considered Dr. Perper's less-than-definitive assertion, I note that the two board certified pulmonologists who considered Mr. Anderson's case, Dr. Fino and Dr. Respher, also presented several medical studies to support their belief that centrilobular emphysema was not caused in part by coal dust exposure. Thus, at best, the record is inconclusive at to whether Mr. Anderson had coal dust related centrilobular emphysema.

At least two of Dr. Perper's principal supports for his diagnosis of "severe" pneumoconiosis are undermined and weak in terms of both documentation and reasoning. As a result, his conclusion that the severe coal workers' pneumoconiosis hastened Mr. Anderson's death has diminished probative value and does not support a finding that coal workers' pneumoconiosis contributed to his death.

In a well documented medical evaluation, Dr. Naeye opined that Mr. Anderson only had mild pneumoconiosis which would not have contributed to his death. While acknowledging the pathological evidence of pneumoconiosis, Dr. Naeye maintained the slides were not indicative of actual profusion of the lesions in Mr. Anderson's lungs. Instead, based on the radiographic evidence that was predominately negative for pneumoconiosis, Dr. Naeye did not believe the pneumoconiosis was sufficient to cause a measurable, adverse impact on his pulmonary function. Additionally, when he reviewed the lung tissue slides, Dr. Naeye noted the mild, simple coal workers' pneumoconiosis lesions were located in areas unrelated to pulmonary function. Further, the amount of focal emphysema associated with coal dust exposure was less than one percent of total emphysema would have had no impact. Even the remaining amount of centrilobular emphysema was not sufficiently severe to cause abnormal pulmonary function. According to medical studies, the centrilobular emphysema is also unrelated to coal dust exposure. According to Dr. Naeye, Mr. Anderson's breathing problems were due to idiopathic, severe pulmonary arterial hypertrophy which led to progressive heart failure.

In assessing Dr. Naeye's medical opinion on remand, I continue to give it diminished probative weight because he stills fails to reconcile his diagnosis of "mild" pneumoconiosis based on his review of the slides with the other pathological evidence provided by Dr. Harlan's gross autopsy findings which indicate a moderate amount of anthracotic nodules throughout the lung tissue upon sectioning. I am also un-persuaded by Dr. Naeye's implicit premise that radiographic evidence of pneumoconiosis must be present before pneumoconiosis may be considered sufficiently severe to have contributed to death. While recognizing Mr. Anderson's severe pulmonary decline in the last few months of his life, Dr. Naeye did not seem to integrate into his assessment Dr. Peacock's clinical observations of pulmonary difficulties associated with

[19]As set out in the initial Decision and Order, only one of the seventeen chest x-rays was positive for pneumoconiosis. That sole, uncontested, positive interpretation was made by a physician who was not a dual qualified radiologist.

both obstructive lung disease and interstitial fibrosis. As directed by the BRB, I have considered Dr. Naeye's finding that idiopathic pulmonary arterial hypertrophy was the major contributing factor in Mr. Anderson's death. In that regard, I simply note that Dr. Naeye's diagnosis of pulmonary arterial hypertension does not preclude a finding the coal workers' pneumoconiosis in Mr. Anderson's lung may also have hastened his death.

Dr. Fino also presented a well documented medical assessment that coal workers' pneumoconiosis did not contribute to Mr. Anderson's death. Based on his extensive review of the medical record and pathology reports, Dr. Fino concluded the limited scope of the autopsy precluded a determination as to the cause of death even though the pathology reports showed the presence of both pneumoconiosis and focal emphysema related to coal dust inhalation,. Nevertheless, Dr. Fino was able exclude pneumoconiosis as a contributing cause of death factor for two reasons. First, based on Mr. Anderson's clinical symptoms, Dr. Fino concluded Mr. Anderson's COPD, with its associated adverse pulmonary impact, was due solely to cigarette smoking. Dr. Fino eliminated coal dust inhalation as a possible etiology because a coal dust-related pulmonary obstruction only occurs when severe pulmonary fibrosis is present and the respiratory impairment has both obstructive and restrictive components. Second, and closely related, coal dust-related emphysema becomes a significant pulmonary factor only in the presence of significantly increasing pneumoconiosis.

In my first evaluation of Dr. Fino's opinion, I found his focus on the clinical symptoms of medical, to the exclusion of legal, pneumoconiosis diminished the probative value of his opinion. Dr. Fino's additional 2005 statement noting that coal dust-related emphysema would become a factor only with increasing significant pneumoconiosis does little to change my mind. Essentially, by focusing on whether Mr. Anderson was totally disabled due to pneumoconiosis and had severe pulmonary fibrosis, Dr. Fino implicitly establishes severe interstitial fibrosis due to coal dust exposure as a predicate for determining whether pneumoconiosis hastened a miner's death. While such a requirement may be medically sound, the court's mandate for a specific mechanism associated with the hastening of death does not raise the evidentiary bar so high that a miner must be totally disabled or have profound coal workers' pneumoconiosis before its presence can be considered to have hastened his death. I have considered the BRB's admonition to reconsider Dr. Fino's analysis of the clinical symptoms since I did not find the presence of legal pneumoconiosis.[20] However, my probative value finding rests principally on his apparent predicate of total disability due to coal workers' pneumoconiosis for a finding that coal workers' pneumoconiosis hastens death.

Finally, Dr. Repsher also presented a well documented opinion that coal workers' pneumoconiosis did not contribute to Mr. Anderson's death. Since the radiographic evidence was predominantly negative for pneumoconiosis and the pathology only revealed the presence of mild pneumoconiosis, Dr. Repsher opined the pneumoconiosis affected only small percentage of Mr. Anderson's lungs. As a result, its presence would not have caused any measurable impairment, or, by implication, had impact on his death.

[20]Since the pathology reports clearly demonstrated the presence of both coal workers' pneumoconiosis and coal dust-related emphysema, an additional inquiry in the first decision about the presence of legal pneumoconiosis was not necessary.

Although less strident on the use of clinical pneumoconiosis in his analysis, and while perhaps the best reasoned discussion in terms supporting medical studies, Dr. Repsher's opinion has a reasoning shortfall similar to Dr. Naeye's evaluation because he fails to discuss Dr. Harlan's gross autopsy of moderate anthracotic lesions throughout the lungs. In other words, Dr. Repsher relies on negative chest x-ray evidence and "mild" pneumoconiosis in the lung tissue samples without considering the additional autopsy observations of Dr. Harlan.

In conclusion, while all of the doctors who considered the role coal workers' pneumoconiosis played in Mr. Anderson's death based their assessments on extensive documentation, due to various reasoning flaws each opinion has diminished probative value. Of particular concern for Mrs. Anderson's survivor claim, the three opinions of Dr. Morse, Dr. Peacock and Dr. Perper are insufficient to establish that coal workers' pneumoconiosis hastened her husband's death under the standard mandated by *Williams*. As result, Mrs. Anderson has failed carry her burden of proof in established that the coal workers' pneumoconiosis in her husband's lung was a substantially contributing cause of his death. Accordingly, Mrs. Anderson's claim for survivor benefits must be denied.

ORDER

The claim of MRS. RACHEL M. ANDERSON for survivor benefits under the Act is **DENIED**.

SO ORDERED:

A

RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: December 19, 2005
Washington, D.C.

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. See 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. See 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board. After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed. At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. See 20 C.F.R. § 725.481. If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).